

eggs with recombinant dicalcin reduced the number of sperm that bound to VE as well as the efficiency of fertilization. By contrast, suppression of intrinsic dicalcin by preincubation with anti-dicalcin antibody increased both efficiencies. Furthermore, in our unique penetration assay, recombinant dicalcin inhibited sperm-VE penetration significantly. To further characterize this observation, we investigated the action of dicalcin on sugar distribution within the VE. Exogenously applied dicalcin increased *in vivo* lectin-reactivity in the VE, accompanied by an increase in the lectin-reactivity of isolated glycoprotein. These results suggested that dicalcin binds to VE glycoproteins and alters the pattern of sugar presentation, and thereby inhibits sperm-egg interaction.

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Program/Abstract # 346**Roles of hypoxic response genes in *Drosophila* primordial germ cell development**

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The cellular responses that allow a cell to survive and adapt to hypoxic stress (low oxygen) are largely conserved. The Hypoxia Inducible Factor transcription factors (HIFs) are the primary transcription factors mediating responses to hypoxic stress. HIFs are composed of alpha and beta subunits. HIFalpha is only stable in hypoxic conditions. The pathway for oxygen-dependent degradation of HIFalpha includes a prolyl-hydroxylase (PHD) and the VHL E3 ligase. The *Drosophila* homologs of HIFalpha, HIFbeta, PHD, and VHL are encoded by the *similar*, *tango*, *fatiga*/*Hph*, and *Vhl* genes, respectively. Previous studies have demonstrated that *similar* has roles in *Drosophila* tracheal development as well as border cell migration. Our research team utilizes *Drosophila* germ cell development as a model for studying cell migration and programmed cell death. The working hypothesis for this project is that primordial germ cell development will be responsive to hypoxic stress. Initial results using wild-type embryos have shown that exposure to hypoxia impacts germ cell migration and/or programmed cell death. We are following up these observations to determine if the key components of the hypoxic response pathway are necessary for germ cell migration and programmed cell death during *Drosophila* development.

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Program/Abstract # 347**A crucial role for lipid phosphorylation in WntD-mediated primordial germ cell migration**

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Signaling pathways such as those controlled by the Wnt family of secreted ligands control many aspects of embryonic development and adult homeostasis. Often, the same pathway is used repeatedly to accomplish different tasks, raising the question of how these pathways are regulated in different biological contexts. Significantly more is known about β -catenin-dependent mechanisms of Wnt signaling than β -catenin-independent mechanisms. Therefore, our previous demonstrations that the *Drosophila* ligand WntD utilizes a β -catenin-independent pathway to control both dorsal/ventral patterning and primordial germ cell (PGC) migration led us to examine the mechanism of WntD signal transduction. We undertook a suppressor screen to identify the WntD receptor and other downstream components and discovered that loss of either *CG16708*, a putative ceramide kinase, or *CG31873*, a putative multi-

substrate lipid kinase, suppresses WntD-induced lethal dorsalization. Additionally, embryo homozygous mutant for both genes display a *wntD* mutant-like phenotype in primordial germ cell migration. These and future studies could help reveal a model of how lipid metabolism controls PGC migration.

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Program/Abstract # 348**Prenylation-deficient heterotrimeric G protein gamma subunits reveal GPCR-mediated signaling events in vivo**

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Geranylgeranylation is a post-translational process involving addition of a polyunsaturated lipid to proteins containing a carboxy-terminal CaaX motif. One family of geranylgeranylated proteins, the heterotrimeric G protein gamma (γ) subunits, is essential for the transduction of signals emanating from G protein coupled receptors (GPCR). To better understand the GPCR-mediated events involved in zebrafish development, we investigated the ability of γ subunits with mutated CaaX motifs to disrupt heterotrimeric G protein signaling when expressed in developing larvae. Our studies reveal that prenylation-deficient versions of γ subunits have the ability to disrupt GPCR signaling by altering the subcellular localization of signaling components. The disruption induced by expressing prenylation-deficient γ subunits ubiquitously or in primordial germ cells (PGC) manifests as a loss of directional PGC migration. The majority of γ subunits have the ability to disrupt PGC migration in the prenylation-deficient form, but only a distinct subset of wild type γ subunits have the ability to reverse this semi-dominant negative effect. To understand the roles that γ protein domains have in contributing to differences in γ signaling capacity in vivo, we constructed γ chimeras with swapped middle, N- or C-terminal domains. Analysis of these chimeras demonstrated that multiple regions and motifs within these regions influence the ability of γ to mediate the signaling pathways necessary for directional PGC migration. Our study of prenylated proteins involved in signal transduction contributes to the understanding of how lipids mediate development.

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Program/Abstract # 349**Hold on: Females modulate sperm release in *Drosophila melanogaster***

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Among species with internal fertilization, sperm fate is influenced by interactions with the female reproductive tract. The nature of these interactions is likely to be affected by the female's environment and physical condition although the extent of the effect(s) is poorly understood. *Drosophila melanogaster* is a useful model system for examining female influence on sperm fate due to the ease with which the female's environment can be modified as well as the detailed understanding of associated reproductive processes such as gametogenesis, mating and fertilization. A female's ability to regulate the release of sperm from storage sites was examined under two sub-optimal, but commonly experienced, conditions: limited exposure to fresh oviposition sites and increasing age. Females exposed infrequently to fresh oviposition media became sperm depleted at a slower rate than did females exposed frequently to fresh media. Lower rates of depletion corresponded with preferential sperm retention within one type of storage structure, the seminal receptacle.